

Structure and function of the skin
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Bacterial infections

Impetigo

- Impetigo is a superficial bacterial skin infection
- Usually caused by either **Staphylococcus aureus** or **Streptococcus pyogenes**.

Features:

- 1) 'golden', crusted skin lesions typically found around the mouth
- 2) very contagious

Management:

Limited, localised disease:

- 1) **topical fusidic acid** is **first-line**
- 2) topical retapamulin is used **second-line** if fusidic acid has been ineffective or is not tolerated
- 3) **MRSA: Topical mupirocin** (Bactroban) should be used in this situation
(Not susceptible to either fusidic acid or retapamulin)

Extensive disease:

- **oral flucloxacillin**
- **oral erythromycin** if penicillin allergic



Bullous impetigo / staphylococcal scalded skin syndrome

- Rarely **Staphylococcus** releases an exfoliating toxin which acts high up in the epidermis:
 - A) **Toxin A:**
 - ⇒ Causes blistering at the site of infection (**bullous impetigo**).
 - B) **Toxin B**
 - ⇒ Spreads through the body causing more widespread blistering (**staphylococcal scalded skin syndrome, SSSS**).
- **SSSS** is more common in **childhood** with **very low mortality rates**.
- In **adults**: often associated with **renal disease** or **immunosuppression**, and **mortality 50%**.
- Both these toxins cleave desmoglein 1 (a desmosomal protein) so **NO mucosal involvement** (this is analogous to pemphigus foliaceus which has the same target antigen)
- SSSS can mimic toxic epidermal necrolysis (TEN) on clinical grounds but can be differentiated in 2 ways:
 - 1) **No mucosal involvement** in SSSS, and
 - 2) **skin biopsy**: a frozen section shows:
 - ⇒ a superficial intraepidermal split in **SSSS**
 - ⇒ It is deeper subepidermal in **TEN**.

TTT:

- Both bullous impetigo and SSSS are treated with antistaphylococcal antibiotics (e.g. **flucloxacillin**) and supportive care.

Cellulitis

- A term used to describe an inflammation of the **skin and subcutaneous tissues**,
- **Streptococcus pyogenes** and **Staphylococcus aureus** are the commonest causative organisms.
- Group B *Streptococcus* has a predilection for diabetic patients.

Features:

- 1) commonly occurs on the shins
- 2) erythema, pain, swelling
- 3) there may be some associated systemic upset such as fever

Management:

- 1) The BNF recommends **flucloxacillin** as **first-line treatment** for mild/moderate cellulitis.
- 2) **Clarithromycin** or **clindamycin** is recommended in **patients allergic to penicillin**.
- 3) Many local protocols now suggest the use of oral clindamycin in patients who have failed to respond to flucloxacillin.
- 4) **Severe cellulitis** should be treated with **intravenous benzylpenicillin + flucloxacillin**.



Ascending cellulitis

Staphylococcus aureus and *Streptococci* are the commonest causative organisms.

Group B *Streptococcus* has a predilection for diabetic patients and is the likeliest causative organism in this scenario.

Ecthyma

- An infection due to **Streptococcus** or **Staphylococcus aureus** or occasionally both.
- It presents as chronic well-demarcated, deeply ulcerative lesions sometimes with an exudative crust.
- It is commoner in developing countries, being associated with poor nutrition and hygiene.
- It is rare in the UK but is seen more commonly in intravenous drug abusers and people with HIV.

Treatment:

⇒ **Phenoxymethylpenicillin** (penicillin V) & **flucloxacillin** (both 500mg 4time/day) 10-14 days



Folliculitis

- Inflammation of the hair follicle.
- It presents as itchy or tender papules and pustules.
- **Staphylococcus aureus** is frequently implicated.
- It is commoner in humid climates and when occlusive clothes are worn.
- A variant occurs in the beard area (called '**sycosis barbae**'), which is commoner in black Africans. This is probably caused by the ability of shaved hair to grow back into the skin, especially if the hair is naturally curly.
- **Extensive, itchy folliculitis** of the upper trunk and limbs should alert one to the possibility of underlying **HIV** infection.
- Folliculitis following use of hot tubs أحواض المياه الساخنة is due to **Pseudomonas ovale**.

Treatment:

- 1) is with topical antiseptics, **topical antibiotics** (e.g. sodium fusidate) or
 - 2) **Oral antibiotics** (e.g. flucloxacillin 500 mg or erythromycin 500 mg both four times daily for 2–4 weeks)
- =====

Boils (furuncles)

- Boils are a rather more deep-seated infection of the skin,
- Often caused by **Staphylococcus**.
- These can cause painful red swellings.
- They are commoner in teenagers and often recurrent.
- Recurrent boils may rarely occur in DM or in immunosuppression.
- Large boils are sometimes called '**carbuncles**'.
- Swabs should be taken to check antibiotic sensitivity as community acquired MRSA is an increasingly common cause.

Treatment

- 1) oral antibiotics (e.g. erythromycin 500 mg four times daily for 10–14 days)
- 2) Occasionally need incision and drainage.
- 3) Prophylaxis: Antiseptics such as povidone iodine or chlorhexidine (as soap) and using a bath oil can be useful in prophylaxis

Erysipelas

- A **Streptococcus pyogenes** infection of the **deep dermis** and **subcutis**.
- Complications include:
 - 1) sepsis,
 - 2) cerebral abscess and
 - 3) Venous sinus thrombosis.
- Treatment relies upon IV antibiotics such as **benzylpenicillin** and **erythromycin**.
- In penicillin allergic patient a **macrolide** is the drug of choice.
- There is a 10% cross allergy between cephalosporins and penicillins.



Mycobacterial infections

Leprosy جذام

- A granulomatous disease primarily affecting the peripheral nerves and skin.
- Caused by *Mycobacterium leprae*.

Features:

- 1) patches of hypopigmented skin:
⇒ typically affecting the buttocks, face, and extensor surfaces of limbs
- 2) sensory loss

The degree of cell mediated immunity determines the type of leprosy a patient will develop.

Low degree of cell mediated immunity → lepromatous leprosy ('multibacillary')

- **extensive** skin involvement
- **symmetrical** nerve involvement

High degree of cell mediated immunity → tuberculoid leprosy ('paucibacillary')

- **limited** skin disease
- **asymmetric** nerve involvement

Management:

WHO recommended **triple therapy**:

- 1) **Rifampicin**,
- 2) **Dapsone** and
- 3) **Clofazimine**



lepromatous leprosy



tuberculoid leprosy

Skin disorders associated with tuberculosis

- 1) lupus vulgaris (accounts for 50% of cases)
- 2) erythema nodosum
- 3) scarring alopecia
- 4) scrofuloderma: breakdown of skin overlying a tuberculous focus
- 5) verrucosa cutis
- 6) gumma

Lupus vulgaris

- The most common form of cutaneous TB seen in the Indian subcontinent.
- It generally occurs on the face and is common around the nose and mouth.
- The initial lesion is an erythematous flat plaque which gradually becomes elevated and may ulcerate later



Viral Infections

Herpes simplex virus

- There are two strains of the herpes simplex virus (HSV) in humans: **HSV-1** and **HSV-2**.
- Whilst it was previously thought HSV-1 accounted for oral lesions (**cold sores**) and HSV-2 for genital herpes it is now known there is considerable overlap

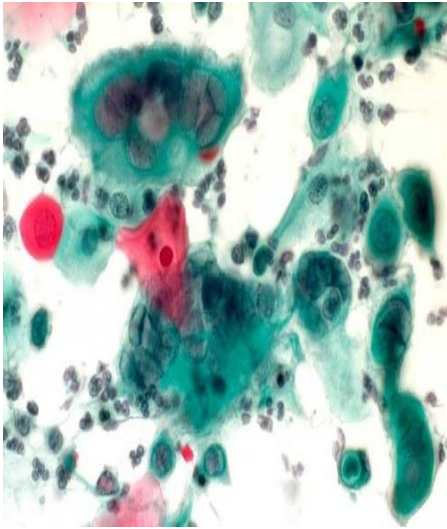
Features:

- 1) Primary infection: may present with a severe gingivostomatitis
- 2) Cold sores
- 3) Painful genital ulceration

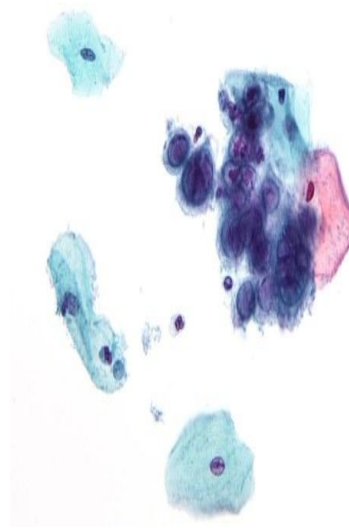


Management:

- 1) **gingivostomatitis**: **oral aciclovir**, chlorhexidine mouthwash
- 2) **cold sores**: topical aciclovir although the evidence base for this is modest
- 3) **Genital herpes**: **oral aciclovir**.
Some patients with frequent exacerbations may benefit from longer term acyclovir



Pap smear.
Multinucleated giant cells representing infection by the herpes simplex virus. Note the 3 M's; Multinucleation, Margination of the chromatin, Molding of the nuclei



Further Pap smear showing the cytopathic effect of HSV (multi-nucleation, ground glass & margined chromatin)

Eczema herpeticum

- Eczema herpeticum describes a severe primary infection of the skin by HSV 1 or 2.
- It is more commonly seen in children with atopic eczema.
- Patients present with new onset of clustered blisters and erosions over an erythematous base.
- The eruption can be widespread and patients may be ill.
- Lesions may be secondarily impetiginised with *Staphylococcus* infection (may need antibiotics).
- Treatment requires **systemic** anti-virals, for example, **aciclovir**.
- Systemic antibiotics may be required if lesions are secondarily impetiginised.
- If life threatening, children should be admitted for **IV acyclovir**



Parvovirus B19

(erythrogenic virus)

- a DNA virus which causes a variety of clinical presentations
- It was identified in the 1980's as the cause of **erythema infectiosum**

Erythema infectiosum: (also known as fifth disease or 'slapped-cheek syndrome')

- most common presenting illness
- systemic symptoms: lethargy, fever, headache
- 'slapped-cheek' rash spreading to proximal arms and extensor surfaces

Other presentations

- 1) asymptomatic
- 2) pancytopenia in immunosuppressed patients
- 3) aplastic crises e.g. in sickle-cell disease
 - ⇒ Parvovirus B19 suppresses erythropoiesis for about a week
 - ⇒ so aplastic anaemia is rare unless there is a chronic haemolytic anaemia

Molluscum contagiosum

المليساء المعدية

- Caused by a **pox DNA virus** infection (Molluscum contagiosum virus)
- It is typically seen in younger children and results in characteristic **small 2-5 mm, pearly, umbilicated lesions**
- Also seen in pt with advanced HIV/AIDS (CD4 < 200 cells/mm³).
- They commonly occur on the face, especially near the eyelids;
- They also occur on genitals and trunk.
- Molluscum contagiosum is **highly infectious**.
- Lesions may be present for up to **12 months** and usually resolve spontaneously.

Management:

- 1) They should be treated with **cryotherapy, liquid nitrogen or curettage**.
- 2) no treatment is recommend in the initial phase due to the benign nature of the condition ???



Genital warts

HPV 6&11

condylomata accuminata

- also known as **condylomata accuminata**
- A common cause of attendance at genitourinary clinics.
- They are caused by the many varieties of the **human papilloma virus HPV**, especially **types 6 & 11**.

Features:

- 1) small (2 - 5 mm) fleshy protuberances which are slightly pigmented
- 2) may bleed or itch



Fig. 23.5 Viral wart.

Management:

- 1) **Topical podophyllum** or **cryotherapy** is commonly used as **first-line treatments** depending on the location and type of lesion:
 - ⇒ **Multiple, non-keratinised warts** are generally best treated with **topical agents**
 - ⇒ **Solitary, keratinised warts** respond better to **cryotherapy**
 - 2) **imiquimod** is a **topical cream** which is generally used **second line**
- ☒ **genital warts:**
- are often **resistant** to treatment and **recurrence** is common
 - although the majority of anogenital infections with HPV clear without intervention within 1-2 years

HPV (**primarily types 16, 18 & 33**) predisposes to **cervical cancer**.

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Pyogenic granuloma (lobular capillary haemangioma)

- A benign vascular lesion of the skin and mucosa.
- The cause is unknown
- The name is a double misnomer - the lesion is neither pyogenic nor a granuloma.
- Usually solitary lesions, appearing as a glistening red papule or nodule that is prone to bleeding and ulceration.
- Lesions often grow rapidly (over weeks), frequently occurring at sites of trauma and commonly involve the digits, arms, head and face.
- Pathologically, it is an inflammatory lesion composed of granulation tissue and chronic inflammatory cells.

Fungal infections

Pityriasis versicolor (tinea versicolor)

- a superficial cutaneous fungal infection caused by *Malassezia furfur* (formerly termed *Pityrosporum ovale*)

Features:

- 1) most commonly affects trunk
- 2) patches may be hypopigmented, pink or brown (**hence versicolor**)
- 3) scale is common
- 4) mild pruritus

Predisposing factors:

- 1) occurs in healthy individuals
- 2) immunosuppression
- 3) malnutrition
- 4) Cushing's

Management:

- 1) topical antifungal e.g. **terbinafine** or **selenium sulphide**
- 2) extensive disease or failure to respond to topical treatment then consider **oral itraconazole**

Tinea

- Tinea is a term given to **dermatophyte fungal infections**.
- Three main types of infection are described depending on what part of the body is infected
 - 1) **Tinea capitis** -----> scalp
 - 2) **Tinea corporis** ----> trunk, legs or arms
 - 3) **Tinea pedis** -----> feet

Tinea capitis (Scalp ringworm)

- a cause of **scarring alopecia** mainly seen in children
- if untreated a raised, pustular, spongy/boggy mass called a kerion may form
- most common cause is **Trichophyton tonsurans** in the UK and the USA
- may also be caused by **Microsporum canis** acquired from cats or dogs

Diagnosis: lesions due to **Microsporum canis** **green fluorescence** under Wood's lamp*. However the most useful investigation is **scalp scrapings**

Management (based on CKS guidelines):

- 1) Oral antifungals:
 - ⇒ **Terbinafine** for **Trichophyton tonsurans** infections and
 - ⇒ **Griseofulvin** for **Microsporum** infections.
- 2) Topical **ketoconazole** shampoo should be given for the first 2 weeks to reduce transmission



Image showing a kerion

*lesions due to **Trichophyton** species do not readily fluoresce under **Wood's lamp**

Tinea corporis

(Ringworm)

- Causes include *Trichophyton rubrum* and *Trichophyton verrucosum* (e.g. From contact with **cattle**)
- Well-defined annular, erythematous lesions with papules and pustules
- May be treated with oral **fluconazole**



Image showing tinea corporis.
Note the well defined border



Image showing tinea corporis

Tinea pedis

(Athlete's foot)

- Characterised by itchy, peeling skin between the toes
- Common in adolescence

Fungal nail infections

(Onychomycosis)

This may be caused by:

- 1) dermatophytes: mainly *Trichophyton rubrum*, accounts for 90% of cases
- 2) yeasts: such as *Candida*
- 3) non-dermatophyte moulds

Features:

- 1) 'unsightly فبيح' nails are a common reason for presentation
- 2) thickened, rough, opaque nails are the most common finding

Investigation:

- 1) nail clippings
- 2) nail scrapings (of the affected)

Management:

- 1) treatment is successful in around 50-80% of people
- 2) diagnosis should be confirmed by microbiology before starting treatment
 - A) **Dermatophyte infection:**
 - 1) *oral terbinafine* is currently recommended **first-line**
 - 2) *Oral itraconazole* as an alternative.
 - 3) fingernail infections → 6 weeks - 3 months therapy is needed
Toenails infections → 3 - 6 months
 - B) **Candida infection:**
 - 1) mild disease should be treated with **topical antifungals** (e.g. Amorolfine)
 - 2) severe infections should be treated with *oral itraconazole* for 12 weeks

Infestations

Scabies

- Scabies is caused by the mite *Sarcoptes scabiei* and is spread by prolonged skin contact.
- It typically affects children and young adults.
- The scabies mite burrows into the skin, laying its eggs in the stratum corneum.
- Intense pruritus associated with scabies is due to a **delayed type IV hypersensitivity reaction to mites/eggs** which occurs about **30 days after the initial infection**.

Features:

- 1) widespread pruritus
- 2) linear burrows on the side of fingers, interdigital webs and flexor aspects of the wrist
- 3) in infants the face and scalp may also be affected
- 4) secondary features are seen due to scratching: excoriation, infection

Scabies may be diagnosed by demonstrating *Sarcoptes scabiei* on **skin scrapings**.

Management:

- 1) **permethrin 5%** is **first-line**
- 2) **malathion 0.5%** is **second-line**
- 3) give appropriate guidance on use (see below)
- 4) pruritus persists for up to 4-6 weeks post eradication

Patient guidance on treatment (from Clinical Knowledge Summaries)

- 1) avoid close physical contact with others until treatment is complete
- 2) all household and close physical contacts should be treated at the same time, even if asymptomatic
- 3) Launder, iron or tumble dry clothing, bedding, towels, etc., on the first day of treatment to kill off mites.

BNF advises to apply the insecticide to all areas, including the face and scalp, contrary to the manufacturer's recommendation.

Patients should be given the following instructions:

- 1) apply the insecticide cream or liquid to cool, dry skin
- 2) pay close attention to areas between fingers and toes, under nails, armpit area, creases of the skin such as at the wrist and elbow
- 3) allow to dry and leave on the skin for 8-12 hours for permethrin, or for 24 hours for malathion, before washing off
- 4) reapply if insecticide is removed during the treatment period, e.g. If wash hands, change nappy, etc
- 5) repeat treatment 7 days later

Crusted (Norwegian) scabies

- Crusted scabies is seen in patients with suppressed immunity, especially HIV.
- The crusted skin will be teeming with hundreds of thousands of organisms.



Management:

- 1) **Ivermectin** is the treatment of choice
- 2) isolation is essential

Papulo-squamous/inflammatory rashes
 Eczema
 Psoriasis
 Urticaria/angio-oedema
 Pityriasis rosea
 Lichen planus
 Granuloma annulare
 Lichen sclerosus

Eczema

Classification of eczema

Endogenous	Exogenous
• Atopic eczema • Discoid eczema • Hand eczema • Seborrhoeic eczema • Venous ('gravitational') eczema • Asteatotic eczema	• Contact eczema – irritant • Contact eczema – allergic • Photosensitive eczema • Lichen simplex/nodular prurigo

Eczema diagnosis:

UK Working Party Diagnostic Criteria for Atopic Eczema:

- 1) An itchy skin condition in the last 12 months
- 2) Plus 3 or more of:
 - 1) onset below age 2 years*
 - 2) history of generally dry skin
 - 3) personal history of other atopic disease***
 - 4) history of flexural involvement**
 - 5) visible flexural dermatitis

*not used in children under 4 years

**or dermatitis on the cheeks and/or extensor areas in children aged 18 months or under

***in children aged under 4 years, history of atopic disease in a first degree relative may be included

Topical steroids

Use weakest steroid cream which controls patient's symptoms

The table below shows topical steroids by potency

Mild	Moderate	Potent	Very potent
Hydrocortisone 0.5-2.5%	Clobetasone butyrate 0.05% (Eumovate)	Fluticasone propionate 0.05% (Cutivate)	Clobetasol propionate 0.05% (Dermovate)
	Betamethasone valerate 0.025% (Betnovate RD)	Betamethasone valerate 0.1% (Betnovate)	

Finger tip rule:

- 1 finger tip unit (FTU) = 0.5 g, sufficient to treat a skin area about twice that of the flat of an adult hand

Topical steroid doses for eczema in adults

Area of skin	Fingertip units per dose
Hand and fingers (front and back)	1.0
A foot (all over)	2.0
Front of chest and abdomen	7.0
Back and buttocks	7.0
Face and neck	2.5
An entire arm and hand	4.0
An entire leg and foot	8.0

The BNF makes recommendation on the quantity of topical steroids that should be prescribed for an adult for a single daily application for 2 weeks:

Area	Amount
Face and neck	15 to 30 g
Both hands	15 to 30 g
Scalp	15 to 30 g
Groin and genitalia	15 to 30 g
Both arms	30 to 60 g
Both legs	100 g
Trunk	100 g

Pompholyx (dyshidrotic eczema)

- A type of eczema which affects both the hands (cheiropompholyx) and the feet (pedopompholyx).

Features:

- 1) small blisters on the palms and soles
- 2) pruritic, sometimes burning sensation
- 3) once blisters burst skin may become dry and crack

Management:

- 1) cool compresses
- 2) emollients
- 3) topical steroids

Psoriasis

- Psoriasis is a common and **chronic skin disorder**. (Prevalence around 2%)
- It generally presents with **red, scaly** patches on the skin
- Patients with psoriasis are at increased risk of **arthritis** and **cardiovascular disease**.

Pathophysiology:

Multifactorial and not yet fully understood

1) genetic:

- Associated HLA-B13, -B17, and -Cw6.
- Strong concordance (70%) in identical twins

2) immunological:

- Abnormal T cell activity stimulates keratinocyte proliferation.
- There is increasing evidence this may be mediated by a novel group of T helper cells producing IL-17, designated Th17.
- These cells seem to be a third T-effector cell subset in addition to Th1 and Th2

3) environmental: it is recognised that psoriasis may be:

- ⇒ triggered by Streptococcal infection
- ⇒ Worsened by Skin trauma, stress,
- ⇒ improved by Sunlight

Recognised subtypes of psoriasis

Plaque psoriasis:

- **the most common** sub-type
- resulting in the typical **well demarcated red, scaly patches**
- affecting the **extensor surfaces, sacrum** and **scalp**

Flexural psoriasis:

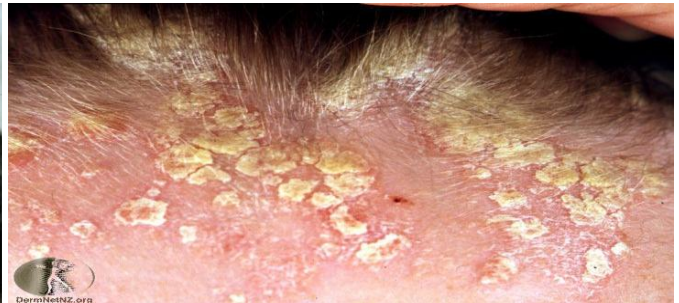
- In contrast to plaque psoriasis **the skin is smooth**

Guttate psoriasis:

- **transient** psoriatic rash
- Frequently triggered by a streptococcal infection.
- Multiple red, teardrop lesions appear on the body

Pustular psoriasis:

- commonly occurs on the palms and soles



This patient presents with typical scalp and hairline psoriasis.

Other features:

- 1) nail signs
- 2) arthritis

Complications:

- 1) Psoriatic arthropathy (around 10%)
- 2) increased incidence of metabolic syndrome
- 3) increased incidence of cardiovascular disease
- 4) increased incidence of venous thromboembolism
- 5) psychological distress

Psoriatic nail changes



- Psoriatic nail changes affect both fingers and toes
- do not reflect the severity of psoriasis but there is an association with psoriatic arthropathy

Nail changes seen in psoriasis:

- 1) pitting
- 2) onycholysis
- 3) subungual hyperkeratosis
- 4) loss of nail

- Psoriasis is mediated by type 1 helper T cells which are involved in the cell mediated response,
- Scalp psoriasis may occur in isolation in patients with no history of psoriasis elsewhere.

Psoriasis exacerbating factors:

The following factors may exacerbate psoriasis or trigger onset:

- 1) trauma, stress
- 2) alcohol
- 3) withdrawal of systemic steroids
- 4) drugs:

- 1) **beta blockers**
- 2) **NSAIDs**
- 3) **lithium,**
- 4) **antimalarials** (chloroquine and hydroxychloroquine),

- Drug-induced psoriasis may occur from less than **one month** to **one year** after the medication is initiated.
- Treatment of drug-induced psoriasis comprises withdrawal of all beta-blocking medications, NSAIDs, antimalarials and lithium, unless absolutely necessary.
- Skin punch biopsy may be performed to exclude other forms of erythroderma or pustulosis.
- Bed rest, bland topical compresses, and low potency topical steroids are useful.
- Frequent emollient use is advisable.
- **Etretinate**, **methotrexate**, or **phototherapy** might be considered.

Psoriasis management:

NICE released guidelines in 2012 on the management of psoriasis and psoriatic arthropathy.

Management of chronic plaque psoriasis:

- 1) regular emollients may help to reduce scale loss and reduce pruritus
- 2) **first-line:**
 - ✓ NICE recommend a **potent corticosteroid** applied once daily plus **vitamin D analogue** applied once daily (applied separately, one in the morning and the other in the evening) for up to 4 weeks as initial treatment
- 3) **second-line:**
 - If no improvement after 8 weeks then
 - ✓ offer a **vitamin D analogue** twice daily
- 4) **third-line:**
 - If no improvement after 8-12 weeks then offer either:
 - ✓ a **potent corticosteroid** applied twice daily for up to 4 weeks
 - or
 - ✓ a **coal tar** preparation applied once or twice daily
- 5) **Short-acting dithranol** can also be used



Using topical steroids in psoriasis

- 1) Topical corticosteroid therapy may lead to skin atrophy, striae and rebound symptoms
- 2) the **face and flexures** are particularly prone to **steroid atrophy** so topical steroids should not be used for more than **1-2 weeks/month**
- 3) Topical steroids are commonly used in flexural psoriasis and there is also a role for mild steroids in facial psoriasis. If steroids are ineffective for these conditions **vitamin D analogues** or **tacrolimus ointment** should be used second line
- 4) **systemic side-effects:**
 - May be seen if potent corticosteroids are used on large areas (> 10% of the body surface area)
- 5) NICE recommend that we aim for a 4 week **break** before starting another course of topical corticosteroids
- 6) they also recommend using:
 - ⇒ potent corticosteroids for no longer than 8 weeks at a time and
 - ⇒ very potent corticosteroids for no longer than 4 weeks at a time
- 7) **Oral-steroids** are contraindicated in psoriasis and although one may see an initial improvement, a very serious rebound effect may be seen.

What should I know about vitamin D analogues?

- 1) examples of vitamin D analogues include:
 - **calcipotriol** (Dovonex),
 - **calcitriol** and **tacalcitol**
- 2) they work by **reducing cell division** and **differentiation**
- 3) adverse effects are uncommon
- 4) unlike corticosteroids they **may be used long-term**
- 5) unlike coal tar and dithranol they **do not smell or stain**
- 6) they tend to **reduce the scale and thickness of plaques** but **not the erythema**
- 7) they should be **avoided in pregnancy**
- 8) the **maximum weekly amount for adults** is **100g**



A 'before and after' image showing the effect of 6 weeks of calcipotriol therapy on a large plaque. Note how the scale has improved but the erythema remains

Scalp psoriasis:

- 1) NICE recommend the use of **potent topical corticosteroids** used **once daily for 4 weeks**
- 2) if no improvement after 4 weeks then either:
 - ⇒ use a **different formulation** of the potent corticosteroid (for example, a shampoo or mousse) **and/or**
 - ⇒ a topical agents to **remove adherent scale** (for example, agents containing salicylic acid, emollients and oils) before application of the potent corticosteroid

Face, flexural and genital psoriasis:

- NICE recommend offering a **mild** or **moderate potency corticosteroid** applied once or twice daily for a **maximum of 2 weeks**
(Topical calcipotriol is usually irritant in flexures)

Secondary care management

A) Phototherapy:

- 1) **Narrow band ultraviolet B light** is now the treatment **of choice**.
If possible this should be given 3 times a week
- 2) **photochemotherapy** is also used - psoralen + ultraviolet A light (PUVA)

Adverse effects:

- skin ageing,
- **squamous cell cancer** (not melanoma)

B) Systemic therapy:

- 1) Oral methotrexate:
 - ✓ Used **first-line**.
 - ✓ It is particularly useful if there is associated joint disease
- 2) ciclosporin
- 3) systemic retinoids
- 4) biological agents: infliximab, etanercept and adalimumab (anti TNF)
- 5) ustekinumab (IL-12 and IL-23 blocker) is showing promise in early trials

Mechanism of action of commonly used drugs:

- 1) coal tar: probably inhibit DNA synthesis
- 2) dithranol: inhibits DNA synthesis, wash off after 30 mins,
SE: burning, staining
- 3) calcipotriol: vitamin D analogue which reduces epidermal proliferation and restores a normal horny layer

Psoriasis can have a significant psychosocial effect on the patient. Control of the disease is incredibly important to maintain a normal life for psoriasis sufferers. Biological immune modifying agents licensed to treat moderate to severe psoriasis, include TNF alpha-blockers (e.g. etanercept, infliximab, adalimumab) and blockers of interleukin 12/23 (i.e. Ustekinumab).

Treatment for psoriasis can be grouped into topical or systemic:

Mild to moderate disease can be controlled via topical treatments such as:

- coal tar
- topical steroids
- topical tacrolimus, and
- phototherapy.

Systemic treatments include:

- methotrexate
- cyclosporin, and
- biological immune modifying agents (as listed above).

Guttate Psoriasis

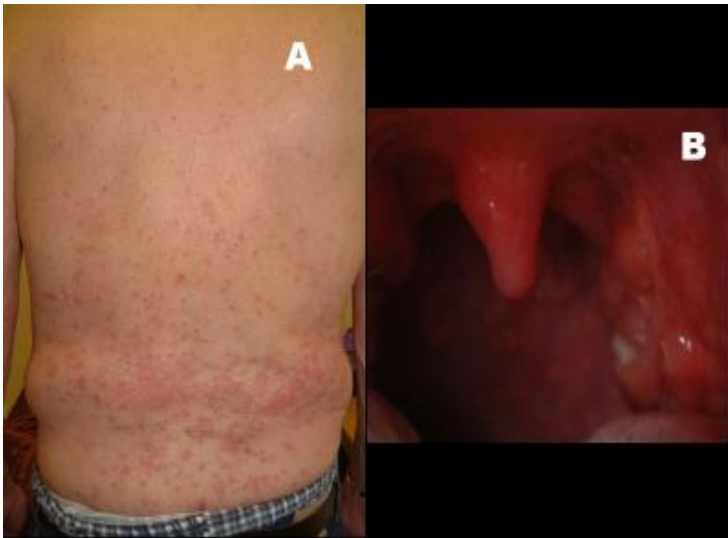
- More common in children and adolescents.
- It may be precipitated by a **streptococcal infection** 2-4 weeks prior to lesions appearing

Features:

- Red scaly tear drop papules on the trunk and limbs

Management:

- 1) most cases resolve spontaneously within 2-3 months
- 2) **NO** firm evidence to support the use of antibiotics to eradicate strept. infection
- 3) topical agents as per psoriasis
- 4) UVB phototherapy
- 5) tonsillectomy may be necessary with recurrent episodes



Pityriasis rosea

- cause unknown, Human herpes virus 7 (HHV-7) a possibility
- tends to affect young adults

Features:

- 1) **herald patch** (usually on trunk)
- 2) Followed 1-2 weeks later by erythematous, oval, scaly (fine scale confined to the outer aspects of the lesions) patches which follow a characteristic distribution with the longitudinal diameters of the oval lesions running parallel to the line of Langer. This may produce a '**fir-tree**' appearance

There is a history of a herald patch, which presents as a single large erythematous plaque on the trunk. This is followed by multiple erythematous plaques along the rib lines on the chest and abdomen, within a few weeks of the herald patch.

Management:

- self-limiting, usually disappears after 4-6 weeks



On the left a typical herald patch is seen. After a few days a more generalised 'fir-tree' rash appears



Differentiating guttate psoriasis and pityriasis rosea

	Guttate psoriasis	Pityriasis rosea
Prodrome	Classically preceded by a streptococcal sore throat 2-4 weeks	Many patients report recent respiratory tract infections but this is not common in questions
Appearance	'Tear drop', scaly papules on the trunk and limbs	Herald patch followed 1-2 weeks later by multiple erythematous, slightly raised oval lesions with a fine scale confined to the outer aspects of the lesions. May follow a characteristic distribution with the longitudinal diameters of the oval lesions running parallel to the line of Langer. This may produce a 'fir-tree' appearance
Treatment / natural history	Most cases resolve spontaneously within 2-3 months Topical agents as per psoriasis UVB phototherapy	Self-limiting, resolves after around 6 weeks



Pityriasis rosea

Lichen planus

A skin disorder of unknown aetiology, most probably being immune mediated.

Features:

- 1) **itchy**, papular rash most common on the palms, soles, genitalia and flexor surfaces of arms
- 2) rash often polygonal in shape, '**white-lace**' pattern on the surface (Wickham's striae)
- 3) **oral involvement** in around **50% of patients**
- 4) **Koebner phenomenon** may be seen (new skin lesions appearing at the site of trauma)
- 5) **nails**: thinning of nail plate, longitudinal ridging
- 6) scarring alopecia



Management:

- 1) **topical steroids** are the mainstay of treatment
- 2) extensive lichen planus may require **oral steroids** or **immunosuppression**

Lichenoid drug eruptions - causes:

- 1) gold
- 2) quinine
- 3) thiazides

Lichen sclerosus

- Previously termed lichen sclerosus et atrophicus.
- Inflammatory condition, usually affects the **genitalia**, more common in **elderly females**.
- Lichen sclerosus leads to **atrophy of the epidermis** forming white plaques
- **itching** is prominent
- A biopsy is often performed to exclude other diagnoses

Management:

- 1) topical steroids and emollients
- 2) increased risk of vulval cancer

- Lichen Planus: purple, pruritic, papular, polygonal rash on flexor surfaces. Wickham's striae over surface. Oral involvement common
- Lichen sclerosus: itchy white spots typically seen on the vulva of elderly women

Granuloma annulare

- papular lesions that are often slightly hyperpigmented and depressed centrally
- typically occur on the dorsal surfaces of the hands and feet, and on the extensor aspects of the arms and legs
- A number of associations were proposed to conditions as DM but there is weak evidence

Facial rashes

Acne rosacea

Acne rosacea is a chronic skin disease of unknown aetiology



Fig. 23.21 Rosacea. Papules and pustules on a background erythema. There are no comedones.

Features:

- 1) Typically affects nose, cheeks and forehead
- 2) Flushing is often first symptom
- 3) Telangiectasia are common
- 4) Later develops into persistent erythema with papules and pustules
- 5) Rhinophyma (red bulbous nose, strawberry)
- 6) Ocular involvement: blepharitis, conjunctivitis, keratitis may occur

management:

- 1) **Topical metronidazole** may be used for mild symptoms (i.e. Limited number of papules and pustules, no plaques)
- 2) **More severe disease** is treated with systemic antibiotics e.g. **Oxytetracycline**
- 3) Recommend daily application of a **high-factor sunscreen**
- 4) **Camouflage creams** may help conceal redness
- 5) **Laser therapy** may be appropriate for patients with **prominent telangiectasia**

Acne vulgaris

- A common skin disorder which usually occurs in adolescence.
- It typically affects the face, neck and upper trunk
- Characterised by the obstruction of the **pilosebaceous follicle** with keratin plugs which results in comedones, inflammation and pustules.

Epidemiology:

- Affects around 80-90% of teenagers, 60% of whom seek medical advice
- Acne may also persist beyond adolescence, with **10-15% of females** and **5% of males over 25 years old** being affected

Pathophysiology is multifactorial:

- Follicular epidermal hyperproliferation resulting in the formation of a keratin plug.
- This in turn causes obstruction of the pilosebaceous follicle.
- Activity of sebaceous glands may be controlled by androgen, although levels are often normal in patients with acne
- Colonisation by the **anaerobic** bacterium **Propioni-bacterium acnes**
- Inflammation

Acne vulgaris management:

Acne may be classified into mild, moderate or severe:

Mild: open and closed comedones with or without sparse inflammatory lesions

Moderate acne: widespread non-inflammatory lesions & numerous papules and pustules

Severe acne: extensive inflammatory lesions, may include nodules, pitting, and scarring

A simple step-up management scheme often used in the treatment of acne is as follows:

- 1) **Single topical therapy:** topical retinoids, benzyl peroxide
- 2) **Combination topical therapy:** topical antibiotic, topical retinoid, benzoyl peroxide
- 3) **Oral antibiotics:** e.g. Oxytetracycline, doxycycline.
 - Improvement may not be seen for 3-4 months.
 - Minocycline now considered less appropriate due to the possibility of **irreversible pigmentation**.
 - ☒ Gram negative folliculitis may occur as a complication of long-term antibiotic use
 - **High-dose oral trimethoprim** is effective if this occurs
- 4) Oral isotretinoin: only under specialist supervision
- 5) There is no role for dietary modification in patients with acne

Isotretinoin

- An oral retinoid used in the treatment of severe acne
- 2/3 of patients have a long term remission or cure following a course of oral isotretinoin

Adverse effects:

- 1) Teratogenicity: females should use 2 forms of contraception (e.g. COC pill and condoms)
- 2) Dry skin, eyes and lips: the most common side-effect of isotretinoin
- 3) Nose bleeds (caused by dryness of the nasal mucosa)
- 4) Hair thinning
- 5) Photosensitivity
- 6) Low mood
- 7) Raised triglycerides TG
- 8) Benign intracranial hypertension: should not be combined with tetracyclines for this.

Seborrhoeic dermatitis in adults

- A chronic dermatitis
- Thought to be caused by an inflammatory reaction related to a proliferation of a normal skin inhabitant, a fungus called **malassezia furfur** (formerly known as pityrosporum ovale).
- It is common, affecting around **2% of the general population**

Features:

- 1) Eczematous lesions on the sebum-rich areas:
 - ⇒ Scalp (may cause *dandruff*),
 - ⇒ Periorbital,
 - ⇒ Auricular and
 - ⇒ Nasolabial folds
- 2) Otitis externa and blepharitis may develop



Fig. 23.12 Seborrhoeic eczema affecting the sides of the nose.

Associated conditions include:

- 1) Hiv
- 2) Parkinson's disease

Scalp disease management:

- 1) Over the counter preparations containing **zinc pyrithione** ('head & shoulders') and **tar** ('neutrogena t/gel') are **first-line**
- 2) The preferred **second-line** agent is **ketoconazole (nizoral)**
- 3) **Selenium sulphide** and **topical corticosteroid** may also be useful

Face and body management:

- 1) Topical antifungals: e.g. **Ketoconazole**
 - 2) Topical steroids: best used for short periods
 - 3) Difficult to treat - recurrences are common
- Seborrhoeic keratosis is a benign skin tumour most commonly seen in elderly patients.
 - Although most commonly seen on the head and neck region, the trunk and limbs may also be involved.
 - It presents as a hyperpigmented, sessile or pedunculated, 'stuck-on' papule or nodule.

Cutaneous lupus erythematosus (CLE)

- Skin disease may occur as part of SLE, or present as cutaneous lupus erythematosus without any systemic disease, and with variable chance of progression to SLE, examples;
 - 1) **Discoid lupus erythematosus (DLE)**
 - 2) **Subacute cutaneous lupus erythematosus (SACLE)**
 - 3) **Acute cutaneous lupus erythematosus (ACLE)**,
- ACLE often accompanies flare of systemic disease.
- ACLE presents as diffuse erythema, maculopapular rash, photosensitivity, and oral ulcers, while DLE presents as well defined scaly plaques, which heal with central scarring
- SACLE is ANA positive in 60% patients. However, only 10-15% progress to SLE with moderate disease activity. 80% patients are anti-Ro antibody positive.

Discoid lupus erythematosus

- A benign disorder generally seen in **younger females**
- It very **rarely progresses to sle** (in **less than 5% of cases**).
- Discoid lupus erythematosus is characterised by **follicular keratin plugs**
- Thought to be **autoimmune** in aetiology

Features:

- 1) Well defined erythematous, raised (plaque) rash, sometimes scaly
- 2) May be photosensitive
- 3) More common on face, neck, ears and scalp (sun exposed areas)
- 4) Lesions heal with atrophy, scarring (may cause scarring alopecia), and pigmentation

Management:

- 1) **Topical steroid cream**
- 2) **Oral antimalarials** may be used **second-line** e.g. **Hydroxychloroquine**
- 3) **Avoid sun exposure**



Discoid lupus erythematosus affecting the scalp

Skin disorders associated with sle

- Photosensitive 'butterfly' rash(spares nasolabial folds)
- Discoid lupus
- Alopecia
- Livedo reticularis: net-like rash
- Raynaud's phenomenon



Neonatal lupus



- This infant has neonatal lupus.
- He presents with a characteristic peri-orbital 'raccoon-eyes' rash.
- Other similar pink to red macules, which may have an annular configuration, may be seen on the scalp, face and extremities.
- The mother is usually positive for anti-Ro or anti-La antibodies but may not have overt lupus erythematosus.
- **Congenital heart block** is sequela that may occur in some infants with neonatal lupus, sometimes requiring pace-maker insertion.

Photosensitive skin disorders

Diseases aggravated by sunlight exposure

- 1) Systemic lupus erythematosus,
- 2) Discoid lupus
- 3) Porphyria PCT (not AIP)
- 4) Herpes labialis (cold sores)
- 5) Pellagra
- 6) Xeroderma pigmentosum
- 7) Solar urticaria
- 8) Polymorphic light eruption

Actinic keratoses

- Actinic, or solar, keratoses (AK)
- A **common premalignant skin lesion (10% squamous cell carcinomas)**
- Develops as a consequence of **chronic sun exposure**

Features:

- 1) Presents as a small, scaly, erythematous macule or papule
- 2) May be pink, red, brown or the same colour as the skin
- 3) Typically on sun-exposed areas e.g. **Temples of head**
- 4) Multiple lesions may be present

Management options include:

- 1) Prevention of further risk: e.g. Sun avoidance, sun cream
- 2) **Fluorouracil cream:**
 - ✓ Typically a 2 - 3 week course.
 - ✓ The skin will become red and inflamed
 - ✓ Sometimes topical hydrocortisone is given following fluorouracil(only after 2 wks from fluorouracil start date) to help settle the inflammation
- 3) **Topical diclofenac:**
 - ✓ May be used for mild aks.
 - ✓ Moderate efficacy but much fewer side-effects
- 4) **Topical imiquimod:** trials have shown good efficacy
- 5) **Cryotherapy**
- 6) **Curettage and cautery**

Erythroderma

Erythroderma is a term used when > 95% of the skin is involved in a rash of any kind

Causes of erythroderma:

- 1) eczema
- 2) psoriasis
- 3) drugs e.g. gold
- 4) lymphoma, leukaemia
- 5) idiopathic

Erythrodermic psoriasis

- May result from progression of chronic disease to an exfoliative phase with plaques covering most of the body.
- Associated with mild systemic upset
- More serious form is an acute deterioration.
- This may be triggered by a variety of factors such as withdrawal of systemic steroids.
- Patients need to be admitted to hospital for management



This image shows the generalised erythematous rash seen in patients with erythroderma, sometimes referred to as '**red man syndrome**'



Note the extensive exfoliation seen in this patient

Cutaneous signs of systemic disease

Erythema nodosum

Overview:

- Inflammation of subcutaneous fat
- Typically causes tender, erythematous, nodular lesions
- Histology of these lesions shows a vasculitis of small venules and panniculitis
- Usually occurs over shins, may also occur elsewhere (e.g. Forearms, thighs)
- Usually resolves within 6 weeks
- Lesions heal without scarring
- The commonest cause is streptococcal infection. However, the commonest potentially serious causes (and therefore those that should be excluded first) include sarcoidosis and tuberculosis. A chest x ray is an important investigation to exclude both of these causes.

Causes:

A) Infection:

- 1) **Streptococci**, (most common)
- 2) Tb,
- 3) Brucellosis

B) Systemic disease:

- 1) Sarcoidosis,
- 2) Inflammatory bowel disease,
- 3) Behcet's

C) Malignancy/lymphoma

D) Drugs:

- 1) Penicillins,
- 2) Sulphonamides,
- 3) Combined oral contraceptive pill

E) Pregnancy



Pyoderma gangrenosum

Features:

- 1) Typically on the lower limbs
- 2) Initially small red papule
- 3) Later deep, red, necrotic ulcers with a violaceous border
- 4) May be accompanied systemic symptoms e.g. Fever, myalgia



Causes:

- 1) Idiopathic in 50%
- 2) Inflammatory bowel disease: ulcerative colitis, crohn's
- 3) Primary biliary cirrhosis
- 4) Rheumatoid arthritis, sle
- 5) Myeloproliferative disorders
- 6) Lymphoma, myeloid leukaemias
- 7) Monoclonal gammopathy (iga)

Management:

- 1) The potential for rapid progression is high in most patients and most doctors advocate **oral steroids** as **first-line treatment**
- 2) Other immunosuppressive therapy, for example **ciclosporin** and **infliximab**, have a role in difficult cases

*note whilst pyoderma gangrenosum can occur in dm it is rare and is generally not included in a differential of potential causes

Dermatitis herpetiformis

- An **autoimmune blistering** skin disorder associated with **coeliac disease**.
- It is caused by **deposition of iga in the dermis**.

Features

- Very itchy, vesicular skin lesions on the extensor surfaces (e.g. Elbows, knees, buttocks)

Skin biopsy:

Direct immunofluorescence shows **deposition of iga** in a **granular pattern** in the **upper dermis**

Management:

- 1) Gluten-free diet
- 2) Dapsone



Erythema multiforme

Features:

- 1) **Target lesions**
- 2) Initially seen on the back of the hands / feet before spreading to the torso
- 3) Upper limbs are more commonly affected than the lower limbs
- 4) Pruritus is occasionally seen and is usually mild

If symptoms are severe and involve blistering and mucosal involvement the term **stevens-johnson syndrome** is used.

Causes:

- 1) Herpes simplex virus (the most common cause), orf*
- 2) Idiopathic
- 3) Mycoplasma, streptococcus
- 4) Penicillin, sulphonamides, carbamazepine, allopurinol, NSAIDS, oral contraceptive pill, nevirapine
- 5) Connective tissue disease e.g. SLE
- 6) Sarcoidosis
- 7) Malignancy



*Orf is a skin disease of sheep and goats caused by a parapox virus

Stevens-Johnson syndrome

Stevens-Johnson syndrome severe form of erythema multiforme associated with mucosal involvement and systemic symptoms

Features

- 1) Rash is typically maculopapular with target lesions being characteristic. May develop into vesicles or bullae
- 2) Mucosal involvement
- 3) Systemic symptoms: fever, arthralgia



Causes: (erythema multiforme **نفس اسباب**)

- 1) Idiopathic
- 2) Mycoplasma, Streptococcus
- 3) Herpes simplex virus, Orf
- 4) Penicillin, sulphonamides, carbamazepine, allopurinol, nsaid, oral contraceptive pill
- 5) Connective tissue disease e.g. SLE
- 6) Sarcoidosis
- 7) Malignancy

Toxic epidermal necrolysis (TEN)

- A potentially life-threatening skin disorder
- Most commonly seen secondary to a drug reaction (in particular sulphonamides and anticonvulsants).
- In this condition the skin develops a **scalded appearance** over an extensive area.
- Some authors consider TEN to be the severe end of a spectrum of skin disorders which includes erythema multiforme and Stevens-Johnson syndrome
- Epidermal desquamation involving more than 30% of body surface area.

Features:

- 1) Systemically unwell e.g. Pyrexia, tachycardic
- 2) Positive Nikolsky's sign: the epidermis separates with mild lateral pressure

It is often idiopathic but may be associated with:

- Viral infections
- Leukaemia
- Lymphoma, and
- Drugs (in particular sulphonamides and anticonvulsants).

Drugs known to induce TEN:

- 1) Sulphonamides
- 2) Carbamazepine, phenytoin
- 3) Penicillins
- 4) Allopurinol
- 5) NSAIDS

Management:

- 1) Stop precipitating factor
- 2) Supportive care, often in ICU
- 3) **Intravenous immunoglobulin** has been shown to be effective and is now commonly used **first-line**
- 4) Other treatment options include: **immunosuppressive agents** (ciclosporin and cyclophosphamide), **plasmapheresis**

DRESS (Drug reaction with eosinophilia and systemic symptoms)

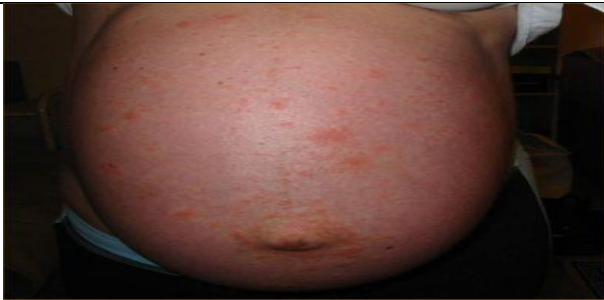
- Severe drug eruption characterised by generalised erythematous rash
- **Often associated with:**
 - 1) Facial oedema,
 - 2) Involvement of Internal organs (liver dysfunction),
 - 3) Hematologic abnormalities (eosinophilia) and
 - 4) Systemic illness (fever).

Fixed drug eruption presents with one or several erythematous-to-dusky patches or plaques with central blister not associated with systemic symptoms and patients are not ill.

Skin disorders associated with pregnancy

Polymorphic eruption of pregnancy

- Pruritic condition associated with last trimester
- Lesions often first appear in abdominal striae
- Management depends on severity:
 - ✓ Emollients, mild potency topical steroids and
 - ✓ Oral steroids may be used



Polymorphic eruption of pregnancy

Pemphigoid gestationis

- Pruritic blistering lesions
- Develop in peri-umbilical region, later spreading to the trunk, back, buttocks and arms
- Usually presents 2nd or 3rd trimester and is rarely seen in the first pregnancy
- Oral corticosteroids are usually required



Acanthosis nigricans

Describes symmetrical, brown, velvety plaques that are often found on the neck, axilla and groin

Causes:

- 1) GIT cancer (Gastric Adinocarcinoma)
- 2) Diabetes mellitus
- 3) Obesity
- 4) Polycystic ovarian syndrome
- 5) Acromegaly
- 6) Cushing's disease
- 7) Hypothyroidism
- 8) Familial
- 9) Prader-Willi syndrome
- 10) Drugs:

- ✓ Oral contraceptive pill,
- ✓ Nicotinic acid



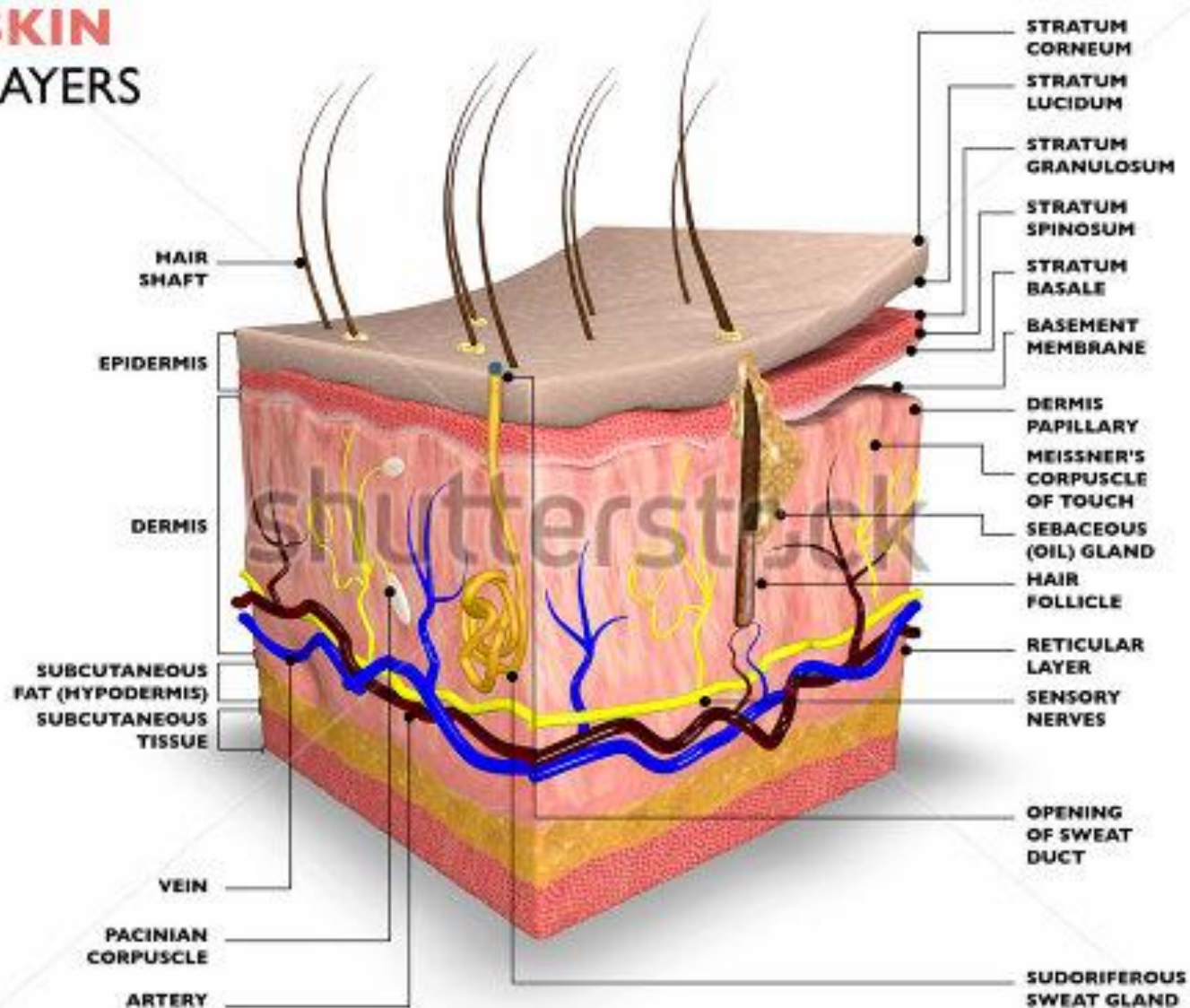
Erythrasma

- Asymptomatic, flat, slightly scaly, pink or brown rash usually found in the groin or axillae
- It is caused by an overgrowth of the diphtheroid *Corynebacterium minutissimum*
- Examination with Wood's light reveals a coral-red fluorescence.
- Topical miconazole or antibacterial are usually effective.
- Oral erythromycin may be used for more extensive infection



(a) (b)
Fig. 23.3 Erythrasma of the axilla, showing pink fluorescence under a Wood's lamp.

SKIN LAYERS



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Bullous disorders

Causes of skin bullae

- 1) Congenital: epidermolysis bullosa
- 2) Autoimmune: bullous pemphigoid, pemphigus
- 3) Insect bite
- 4) Trauma/friction
- 5) Drugs: barbiturates, furosemide

Bullous pemphigoid

- An autoimmune condition causing **sub-epidermal blistering** of the skin.
- Pemphigoid is a disease of the elderly (over 60 years) characterised by the development of large blisters that heal without scarring.
- The condition is caused by immunoglobulin (igg) autoantibodies against components of the basement membrane.(**antibodies against hemidesmosomal proteins** BP180 and BP230)
- Blistering in pemphigoid occurs at the **subepidermal level** - deeper than the blisters of pemphigus vulgaris (which occur at the dermal-epidermal junction); hence the tense blisters seen in pemphigoid.
- Bullous pemphigoid results from the presence of autoantibodies that induce disruption of the basement membrane zone. This disruption leads to the formation of subepidermal blisters.
- In pemphigus Blisters are thin-walled and fragile - few intact blisters are ever seen.
- In pemphigus vulgaris, mucous membrane involvement is more common, and intact bullae are rare.

Features include

- 1) **Itchy, tense** blisters typically **around flexures**
- 2) The blisters usually **heal without scarring**
- 3) **Mouth is usually spared***

Skin biopsy for routine and direct immunofluorescence:

- ☒ Skin biopsy will reveal a **subepidermal blister** with an **infiltrate of eosinophils**.
- ☒ Immunofluorescence: shows deposition of **igg** and complement (**C 3**) at the **basement membrane zone** (dermo-epidermal junction).

Management:

- 1) Referral to dermatologist for biopsy and confirmation of diagnosis
- 2) **Oral corticosteroids** are the **mainstay of treatment**
- 3) **Topical corticosteroids, immunosuppressants** and **antibiotics** are also used

*in reality around 10-50% of patients have a degree of mucosal involvement. It would however be unusual for an exam question to mention mucosal involvement as it is seen as a classic differentiating feature between pemphigoid and pemphigus.



Pemphigus vulgaris

- Pemphigus vulgaris is an **autoimmune disease**
- It is more common in the **Ashkenazi Jewish population**
- Thin-walled blisters that rupture easily. (intact blisters are rarely seen)
- Caused by **antibodies directed against desmoglein 3**, a cadherin-type epithelial cell adhesion molecule

(exfoliatin toxin of Staph a target **desmoglein 1**...تذكر)

Features:

- 1) **Mucosal ulceration** is common and often the presenting symptom.
- 2) **Oral involvement** is seen in 50-70% of patients
- 3) **Skin blistering:**
 - ✓ Flaccid, easily ruptured vesicles and bullae.
 - ✓ Lesions are typically **painful** but **not itchy**.
 - ✓ These may develop months after the initial mucosal symptoms.
 - ✓ **Nikolsky's** describes the spread of bullae following application of horizontal, tangential pressure to the skin
- 4) Acantholysis on biopsy Immunofluorescent shows deposition of immunoglobulin igg **within the epidermis** directed against intercellular cement, resulting in a '**chicken wire**' appearance

Intraepidermal blister with **acantholytic cells** (cells which have lost their intercellular connections) is seen in the pemphigus group of disease (pemphigus vulgaris and pemphigus foliaceus).



Management:

- 1) **High-dose corticosteroids.**
- 2) **Immunosuppressants**

Skin tumours

Benign cutaneous tumours

Potentially pre-malignant cutaneous tumours

Malignant cutaneous tumours

Keratoacanthoma (KA)

- A benign epithelial tumour of pilosebaceous gland. (**low grade malignant**),
- More common in males
- They are more frequent in middle age and do not become more common in old age (unlike basal cell and squamous cell carcinoma)
- Some experts support classifying KA as a variant of invasive SCC.

Features - said to look like a volcano or crater فوهة البركان

- 1) Initially a **smooth dome-shaped papule**
 - 2) Rapidly grows over few weeks to months to become a **crater centrally-filled with keratin**
 - 3) **Spontaneous regression** of keratoacanthoma within **3-6 months**, often resulting in a **scar**.
- Such lesions **should however be urgently excised** as it is difficult clinically to exclude squamous cell carcinoma. Removal also may prevent scarring.



- Seborrheic keratosis is a benign skin tumour most commonly seen in elderly patients.
- Although most commonly seen on the head and neck region, the trunk and limbs may also be involved.
- It presents as a hyperpigmented, sessile or pedunculated, 'stuck-on' papule or nodule.

Actinic keratoses

- Actinic, or solar, keratoses (AK)
- A **common premalignant skin lesion (10% squamous cell carcinomas)**
- Develops as a consequence of **chronic sun exposure**

Features:

- 1) Presents as a small, scaly, erythematous macule or papule
- 2) May be pink, red, brown or the same colour as the skin
- 3) Typically on sun-exposed areas e.g. **Temples of head**
- 4) Multiple lesions may be present

Management options include:

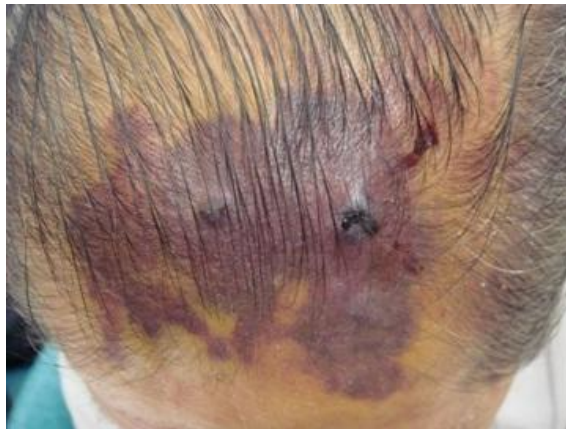
- 1) Prevention of further risk: e.g. Sun avoidance, sun cream
- 2) **Fluorouracil cream:**
 - ✓ Typically a 2 - 3 week course.
 - ✓ The skin will become red and inflamed
 - ✓ Sometimes topical hydrocortisone is given following fluorouracil(only after 2 wks from fluorouracil start date) to help settle the inflammation
- 3) **Topical diclofenac:**
 - ✓ May be used for mild aks.
 - ✓ Moderate efficacy but much fewer side-effects
- 4) **Topical imiquimod:** trials have shown good efficacy
- 5) **Cryotherapy**
- 6) **Curettage and cautery**

Basal cell carcinoma



- This patient presents with a **rodent ulcer** with **rolled pearly edges** and **small telangiectasias**.
- It is the commonest malignant skin tumour and most commonly occurs in elderly patients with sun-damaged skin.

Angiosarcoma



- A malignant vascular tumour seen most commonly in elderly males.
- It most commonly affects the scalp and forehead
- Presents with an infiltrative vascular patch or plaque with super-imposed nodules which may bleed with minor trauma.
- They have a poor prognosis.
- Angiosarcomas can also occur in areas of chronic lymphoedema.

-
- **Pyogenic granuloma** is a benign vascular tumour that presents as a small, bright red, sessile or pedunculated papule.
 - It can occur at sites of previous trauma.

- Senile purpuras commonly occur on the arms of elderly patients and usually resolve after a few weeks, only to recur on minor trauma.

Malignant melanoma

Prognostic factors

- ☒ **The invasion depth of a tumour** (Breslow depth) is the single most important factor in determining prognosis of patients with malignant melanoma

Breslow Thickness	Approximate 5 year survival
< 1 mm	95-100%
1 - 2 mm	80-96%
2.1 - 4 mm	60-75%
> 4 mm	50%

Lentigo maligna

- Lentigo maligna is a type of melanoma in-situ.
- It typically progresses slowly but may at some stage become invasive causing lentigo maligna melanoma.

Disorders of blood vessels/lymphatics

Leg ulcers

Pressure sores

Vasculitis

Lymphatics

Venous ulceration

Venous ulceration is typically seen **above the medial malleolus**

Investigations:

Ankle-brachial pressure index (ABPI)

It is important in non-healing ulcers to assess for poor arterial flow which could impair healing

- ⇒ A 'normal' ABPI may be regarded as between **0.9 - 1.2**.
- ⇒ **Values below 0.9** indicate **arterial disease**.
- ⇒ Interestingly, values above 1.3 may **also indicate arterial disease**, in the form of **false-negative** results secondary to arterial calcification (e.g. In diabetics)

Management:

- 1) **Compression bandaging**, usually 4 layer (only treatment shown to be of real benefit)
- 2) **Oral pentoxifylline**, a peripheral vasodilator, improves healing rate
- 3) Small evidence base supporting use of **flavonoids**
- 4) **Little evidence to suggest benefit from hydrocolloid dressings, topical growth factors, ultrasound therapy and intermittent pneumatic compression**

Pressure ulcers

- The following is based on a 2009 NHS Best Practice Statement.
- Some selected points are listed below. NICE also published guidelines in 2014.
- Pressure ulcers develop in patients who are unable to move parts of their body due to illness, paralysis or advancing age.
- They typically develop over bony prominences such as the sacrum or heel.
- The following factors predispose to the development of pressure ulcers:
 - 1) **Malnourishment**
 - 2) **Incontinence**
 - 3) **Lack of mobility**
 - 4) **Pain** (leads to a reduction in mobility)
- **The Waterlow score** is widely used to screen for patients who are at risk of developing pressure areas.
- It includes a number of factors including BMI, nutritional status, skin type, mobility and continence.
- Grading of pressure ulcers:

The following is taken from the European Pressure Ulcer Advisory Panel classification system.

Grade	Findings
Grade 1	• Non-blanchable erythema of intact skin. • Discolouration of the skin, warmth, oedema, induration or hardness may also be used as indicators, particularly on individuals with darker skin
Grade 2	• Partial thickness skin loss involving epidermis or dermis, or both. • The ulcer is superficial and presents clinically as an abrasion or blister
Grade 3	• Full thickness skin loss involving damage to or necrosis of subcutaneous tissue that may extend down to, but not through, underlying fascia.
Grade 4	• Extensive destruction, tissue necrosis, or damage to muscle, bone or supporting structures with or without full thickness skin loss

Management:

- 1) **A moist wound environment encourages ulcer healing:**
 - ⇒ **Hydrocolloid dressings** and **hydrogels** may help facilitate this.
 - ⇒ The use of **soap should be discouraged** to avoid drying the wound
- 2) **Wound swabs should not be done routinely** as the vast majority of pressure ulcers are colonised with bacteria.
- 3) The decision to use **systemic antibiotics** should be taken on a clinical basis (e.g. **Evidence of surrounding cellulitis**)
- 4) Consider referral to the tissue viability nurse
- 5) Surgical debridement may be beneficial for selected wounds

Disorders of pigmentation

Hypopigmentation

Hyperpigmentation

Vitiligo

- Vitiligo is an autoimmune condition which results in the loss of melanocytes and consequent depigmentation of the skin.
- It is thought to affect around 1% of the population and symptoms typically develop by the age of 20-30 years.

Features:

- 1) Well demarcated patches of depigmented skin
- 2) The peripheries tend to be most affected
- 3) Trauma may precipitate new lesions (Koebner phenomenon)

Associated conditions:

- 1) Type 1 diabetes mellitus
- 2) Addison's disease
- 3) Autoimmune thyroid disorders
- 4) Pernicious anaemia
- 5) Alopecia areata

Management:

- 1) Sun block for affected areas of skin
- 2) Camouflage make-up
- 3) **Topical corticosteroids** may reverse the changes if applied early
- 4) There may also be a role for **topical tacrolimus** and **phototherapy (NBUVB)**, although caution needs to be exercised with light-skinned patients



Onycholysis

Onycholysis describes the separation of the nail plate from the nail bed

Causes:

- 1) Idiopathic
- 2) Trauma e.g. Excessive manicuring
- 3) Infection: especially fungal
- 4) Skin disease: psoriasis, dermatitis
- 5) Impaired peripheral circulation e.g. Raynaud's
- 6) Systemic disease: hyper- and hypothyroidism

Yellow nail syndrome

Slowing of the nail growth leads to the characteristic thickened and discoloured nails seen in yellow nail syndrome.

Associations

- 1) Congenital lymphoedema
- 2) Pleural effusions
- 3) Bronchiectasis
- 4) Chronic sinus infections

Disorders of hair
Hair loss
Increased hair growth

Alopecia

Alopecia may be divided into **scarring** (destruction of hair follicle) and **non-scarring** (preservation of hair follicle)

Scarring alopecia:

- 1) Trauma, burns
- 2) Radiotherapy
- 3) Lichen planus
- 4) Discoid lupus
- 5) Tinea capitis*

Non-scarring alopecia:

- 1) Male-pattern baldness
- 2) Cytotoxic drugs, carbimazole, heparin, oral contraceptive pill, colchicine, Na valproate
- 3) Nutritional: iron and zinc deficiency
- 4) Autoimmune: alopecia areata
- 5) Telogen effluvium (hair loss following stressful period e.g. Surgery)
- 6) Trichotillomania

*scarring may develop in untreated tinea capitis if a kerion develops

Alopecia areata

- Alopecia areata is a presumed autoimmune condition
- Causing localised, well demarcated patches of hair loss.
- At the edge of the hair loss, there may be small, broken 'exclamation mark' hairs
- Hair will regrow in 50% of patients by 1 year, and in 80-90% eventually.
- Careful explanation is therefore sufficient in many patients.
- **Other treatment options include:**
 - 1) Topical or intralesional corticosteroids
 - 2) Topical minoxidil
 - 3) Phototherapy
 - 4) Dithranol
 - 5) Contact immunotherapy
 - 6) Wigs

Hirsutism and hypertrichosis

- Hirsutism is often used to describe **androgen-dependent hair growth in women**
- Hypertrichosis being used for **androgen-independent hair growth**

Hirsutism

Causes of hirsutism:

- Polycystic ovarian syndrome is the most common causes of hirsutism.
- Other causes include:
 - 1) Cushing's syndrome
 - 2) Congenital adrenal hyperplasia CAH
 - 3) Androgen therapy
 - 4) Obesity: due to peripheral conversion oestrogens to androgens
 - 5) Adrenal tumour
 - 6) Androgen secreting ovarian tumour
 - 7) Drugs: phenytoin

Assessment of hirsutism:

- **Ferriman-Gallwey** scoring system: 9 body areas are assigned a score of 0 - 4, a score > 15 is considered to indicate moderate or severe hirsutism

Management of hirsutism:

- 1) Advise weight loss if overweight
- 2) Cosmetic techniques such as waxing/bleaching - not available on the NHS
- 3) Consider using combined oral contraceptive pills such as
 - ✓ Co-cyprindiol (Dianette): should not be used long-term due to the increased risk of **venous thromboembolism**
 - ✓ Ethinylestradiol and drospirenone (Yasmin)
- 4) Facial hirsutism: **topical eflornithine** - contraindicated in **pregnancy and breast-feeding**

Hypertrichosis

Causes of hypertrichosis:

- 1) Drugs:
 - ✓ Minoxidil,
 - ✓ Ciclosporin,
 - ✓ Diazoxide
- 2) Congenital hypertrichosis lanuginosa,
- 3) Congenital hypertrichosis terminalis
- 4) Porphyria cutanea tarda
- 5) Anorexia nervosa

Polycystic ovarian syndrome

- Polycystic ovary syndrome (PCOS) is a complex condition of ovarian dysfunction
- Affect between 5-20% of women of reproductive age
- The aetiology of PCOS is not fully understood.
- Both hyperinsulinaemia and high levels of LH are seen in PCOS and there appears to be some overlap with the metabolic syndrome.

Features:

- 1) Subfertility and infertility.
- 2) Menstrual disturbances: oligomenorrhea and amenorrhoea.
- 3) Hirsutism, acne (due to hyperandrogenism).
- 4) Obesity.
- 5) Acanthosis nigricans (due to insulin resistance).

Investigations:

- 1) Pelvic ultrasound: multiple cysts on the ovaries
- 2) FSH, LH, prolactin, TSH, and testosterone are useful investigations:
 - Raised LH: FSH ratio is a 'classical' feature but is no longer thought to be useful in diagnosis.
 - Prolactin may be normal or mildly elevated.
 - Testosterone may be normal or mildly elevated - however, if markedly raised consider other causes
- 3) Check for impaired glucose tolerance

Polycystic ovarian syndrome management:

- Management is complicated because the aetiology of PCOS is not fully understood.

1) General:

- Weight reduction if appropriate
- If a women requires contraception then a combined oral contraceptive (COC) pill may help regulate her cycle and induce a monthly bleed (see below)

2) Hirsutism and acne:

- **A COC pill** may be used help manage hirsutism.
- Possible options include:
 - ✓ **A third generation COC** which has fewer androgenic effects or
 - ✓ **Co-cyprindiol** which has an anti-androgen action.Both of these types of COC may carry an **increased risk of venous thromboembolism**
- If doesn't respond to COC then **topical eflornithine** may be tried
- **Spironolactone**, **flutamide** and **finasteride** may be used under specialist supervision

3) Infertility:

- Weight reduction if appropriate
- The management of infertility in patients with PCOS should be supervised by a specialist.
- There is an ongoing debate as to whether metformin, clomifene or a combination should be used to stimulate ovulation.

A) Clomifene:

- 1) Work by occupying hypothalamic oestrogen receptors without activating them. This interferes with the binding of oestradiol and thus prevents negative feedback inhibition of FSH secretion
- 2) A 2007 trial published in the NEJM suggested clomifene was the most effective treatment.
- 3) There is a potential risk of multiple pregnancies with anti-oestrogen therapies such as clomifene.

B) Metformin:

- 1) The RCOG published an opinion paper in 2008 and concluded that on current evidence metformin is not a first line treatment of choice in the management of PCOS
- 2) Metformin is also used, either combined with clomifene or alone, particularly in patients who are obese

C) Gonadotrophins

Morphological description of skin lesions

- **Atrophy:** Thinning of the skin
- **Vesicle:** A small fluid-filled blister
- **Bulla:** A large fluid-filled blister
- **Crusted:** Dried serum or exudate on the skin
- **Petechia:** Pinpoint-sized macule of blood in the skin
- **Purpura:** Larger macule or papule of blood in the skin which does not blanch on pressure
- **Ecchymosis:** Large confluent area of purpura ('bruise')
- **Erosion:** Denuded area of skin (partial epidermal loss)
- **Ulcer:** Deeper denuded area of skin (full epidermal and dermal loss)
- **Excoriation:** Scratch mark
- **Fissure:** Deep linear crack or crevice (often in thickened skin)
- **Lichenified:** Thickened epidermis with prominent normal skin markings
- **Macule:** Flat, circumscribed non-palpable lesion
- **Papule:** Small palpable, circumscribed lesion (□ □0.5 cm)
- **Nodule:** Large papule (□ □0.5 cm)
- **Plaque:** Large flat-topped, elevated, palpable lesion
- **Pustule:** Yellowish white pus-filled lesion
- **Scaly:** Visible flaking and shedding of surface skin
- **Telangiectasia:** Abnormal visible dilatation of blood vessels
- **Weal:** Itchy raised 'nettle rash'-like swelling due to dermal oedema

Test Use Clinical example

- Skin swabs Bacterial culture Impetigo
- Blister fluid Electron microscopy and viral culture Herpes simplex
- Skin scrapes Fungal culture
- Microscopy
- Tinea pedis
- Scabies
- Nail sampling Fungal culture Onychomycosis
- Wood's light Fungal fluorescence Scalp ringworm
- Erythrasma
- Blood tests Serology Streptococcal cellulitis
- Autoantibodies Systemic lupus erythematosus
- HLA typing Dermatitis herpetiformis
- DNA analysis Epidermolysis bullosa
- Skin biopsy Histology General diagnosis
- Immunohistochemistry Cutaneous lymphoma
- Immunofluorescence Immunobullous disease
- Culture Mycobacteria/fungi
- Patch tests Allergic contact eczema Hand eczema
- Urine Dipstick (glucose)
- Cytology (red cells)
- Diabetes mellitus
- Vasculitis
- Dermatoscopy (direct microscopy of skin) Assessment of pigmented lesions Malignant melanoma

Allergy tests

Skin prick test	➤ Most commonly used test as easy to perform and inexpensive. ➤ Drops of diluted allergen are placed on the skin after which the skin is pierced using a needle. ➤ A large number of allergens can be tested in one session. ➤ Normally includes a histamine (positive) and sterile water (negative) control. ➤ A wheal will typically develop if a patient has an allergy. ➤ Can be interpreted after 15 minutes ➤ Useful for food allergies and also pollen
Radioallergo sorbent test (RAST)	➤ Determines the amount of ige that reacts specifically with suspected or known allergens, for example ige to egg protein. ➤ Results are given in grades from 0 (negative) to 6 (strongly positive) ➤ Useful for food allergies, inhaled allergens (e.g. Pollen) and wasp/bee venom ➤ Blood tests may be used when skin prick tests are not suitable, for example if there is extensive eczema or if the patient is taking antihistamines
Skin patch testing	➤ Useful for contact dermatitis. ➤ Around 30-40 allergens are placed on the back. Irritants may also be tested for. ➤ The patches are removed 48 hours later with the results being read by a dermatologist after a further 48 hours

Café-au-lait spots

Hyperpigmented lesions that vary in colour from light brown to dark brown, with borders that may be smooth or irregular

Causes

- 1) Neurofibromatosis type I & II
- 2) Tuberous sclerosis
- 3) Fanconi anaemia
- 4) Mccune-Albright syndrome

Contact dermatitis

There are two main types of contact dermatitis

A) **Irritant contact dermatitis:**

- Common - non-allergic reaction due to weak acids or alkalis e.g. Detergents المنظفات
- Often seen on the hands.
- Erythema is typical,
- Crusting and vesicles are rare

B) **Allergic contact dermatitis:**

- Type IV hypersensitivity reaction
- Uncommon
- Often seen on the head following hair dyes
- Presents as an acute weeping eczema which predominately affects the margins of the hairline rather than the hairy scalp itself.
- Topical potent steroid is indicated

Cement is a frequent cause of contact dermatitis. The alkaline nature of cement may cause an irritant contact dermatitis whilst the dichromates in cement also can cause an allergic contact dermatitis.....الاسمنت يعمل الاتنين.....

Nickel dermatitis

- Nickel is a common cause allergic contact dermatitis and is an example of a type IV hypersensitivity reaction.
- It is often caused by jewellery such as watches
- It is diagnosed by a skin patch test

Drugs causing photosensitivity

- Thiazides
- Tetracyclines, sulphonamides, ciprofloxacin
- Sulphonylureas
- Amiodarone
- Nsaids e.g. Piroxicam
- Psoralens

Erythema ab igne

- Erythema ab igne is a skin disorder caused by over exposure to infrared radiation.
- Characteristic features include reticulated, erythematous patches with hyperpigmentation and telangiectasia.
- A typical history would be an elderly woman who always sits next to an open fire.
- If the cause is not treated then patients may go on to develop **squamous cell skin cancer**



Hyperhidrosis

Hyperhidrosis describes the excessive production of sweat

Management options include:

- 1) **Topical aluminium chloride** preparations are first-line. Main side effect is skin irritation
- 2) **Iontophoresis**: particularly useful for patients with palmar, plantar and axillary hyperhidrosis
- 3) **Botulinum toxin**: currently licensed for axillary symptoms
- 4) Surgery: e.g.

Endoscopic transthoracic sympathectomy

- ✓ Patients should be made aware of the risk of compensatory sweating

Koebner phenomenon

The Koebner phenomenon describes skin lesions which appear at the site of injury. It is seen in:

- 1) Psoriasis
- 2) Vitiligo
- 3) Lichen planus
- 4) Lichen sclerosus
- 5) Warts
- 6) Molluscum contagiosum

Myxoid cyst

- Myxoid cysts (also known as mucous cysts)
- Benign ganglion cysts usually found on the distal, dorsal aspect of the finger.
- There is usually osteoarthritis in the surrounding joint.
- They are more common in middle-aged women.

Keloid scars

- Keloid scars are tumour-like lesions that arise from the connective tissue of a scar and extend beyond the dimensions of the original wound

Predisposing factors:

- 1) Ethnicity: more common in people with dark skin
- 2) Occur more commonly in young adults, rare in the elderly
- 3) Common sites (in order of decreasing frequency): sternum, shoulder, neck, face, extensor surface of limbs, trunk

Keloid scars are less likely if incisions are made along relaxed skin tension lines*

Treatment:

- 1) Early keloids may be treated with intra-lesional steroids e.g. Triamcinolone
- 2) Excision is sometimes required

*Langer lines were historically used to determine the optimal incision line. They were based on procedures done on cadavers but have been shown to produce worse cosmetic results than when following skin tension lines

Porphyria cutanea tarda

- Porphyria cutanea tarda is the most common hepatic porphyria.
- It is due to an inherited defect in uroporphyrinogen decarboxylase or caused by hepatocyte damage e.g. Alcohol, HCV, oestrogens, irons

Features:

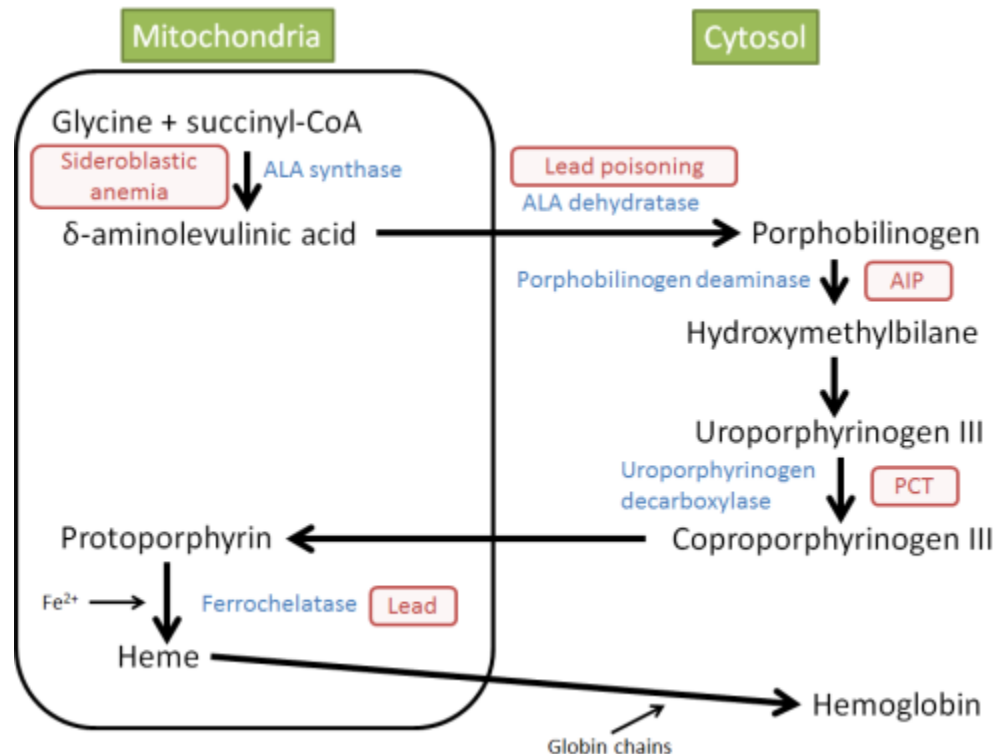
- 1) Classically presents with photosensitive rash with blistering and skin fragility on the face and dorsal aspect of hands (most common feature)
- 2) Hypertrichosis
- 3) Hyperpigmentation

Investigations:

- Urine: elevated uroporphyrinogen and pink fluorescence of urine under Wood's lamp

Management:

- 1) Chloroquine
- 2) Venesection (Hb target 12)



Pruritus

The table below lists the main characteristics of the most important causes of pruritus

Liver disease	• History of alcohol excess • Stigmata of chronic liver disease: spider naevi, bruising, palmar erythema, gynaecomastia etc • Evidence of decompensation: ascites, jaundice, encephalopathy
Iron deficiency anaemia	• Pallor • Other signs: koilonychia, atrophic glossitis, post-cricoid webs, angular stomatitis
Polycythaemia	• Pruritus particularly after warm bath • 'Ruddy complexion' • Gout • Peptic ulcer disease
Chronic kidney disease	• Lethargy & pallor • Oedema & weight gain • Hypertension
Lymphoma	• Night sweats • Lymphadenopathy • Splenomegaly, hepatomegaly • Fatigue

Other causes:

- 1) Hyper- and hypothyroidism
- 2) Diabetes
- 3) Pregnancy
- 4) 'Senile' pruritus
- 5) Urticaria
- 6) Skin disorders: eczema, scabies, psoriasis, pityriasis rosea

Shin lesions

The differential diagnosis of shin lesions includes the following conditions:

- 1) Erythema nodosum
- 2) Pretibial myxoedema
- 3) Pyoderma gangrenosum
- 4) Necrobiosis lipoidica diabeticorum

Below are the characteristic features:

Erythema nodosum:

- Symmetrical, erythematous, tender, nodules which heal without scarring
- Most common causes are strept. Infections, sarcoidosis, Tuberculosis , inflammatory bowel disease and drugs (penicillins, sulphonamides, oral contraceptive pill)

Pretibial myxoedema:

- Symmetrical, erythematous lesions seen in Graves' disease
- Shiny, orange peel skin, raised & indurated

Pyoderma gangrenosum:

- Initially small red papule
- Later deep, red, necrotic ulcers with a violaceous border
- Idiopathic in 50%, may also be seen in inflammatory bowel disease, connective tissue disorders and myeloproliferative disorders

Necrobiosis lipoidica diabeticorum

- Shiny, painless areas of yellow/red skin typically on the shin of diabetics
- Often associated with telangiectasia, Occasionally ulceration of the lesion may occur
- Necrobiosis lipoidica is a disorder of collagen degeneration with a granulomatous response, thickening of blood vessel walls, and fat deposition.
- The cause is unknown, but the leading theory focused on diabetic microangiopathy.
- Necrobiosis is often mistaken for eczema but rather than responding to **steroids may actually deteriorate.** مهم جدا
- It is usually related to diabetes, but can also occur in patients with rheumatoid arthritis. Necrobiosis may precede symptoms and signs of diabetes by several months.



Skin disorders associated with diabetes

Note whilst pyoderma gangrenosum can occur in diabetes mellitus it is rare and is often not included in a differential of potential causes

1) **Necrobiosis lipoidica:**

- Shiny, painless areas of yellow/red/brown skin typically on the shin
- Often associated with surrounding telangiectasia

2) **Infection:**

- Candidiasis
- Staphylococcal

3) **Neuropathic ulcers**

4) **Vitiligo**

5) **Lipoatrophy**

6) **Granuloma annulare:**

- Papular lesions that are often slightly hyperpigmented and depressed centrally
- It is not clear from recent studies if there is actually a significant association between diabetes mellitus and granuloma annulare, but it is often listed in major textbooks



- This is granuloma annulare, the site may well be atypical in diabetic patients.
- The treatment is 'masterful inactivity'.
- The eruption should disappear spontaneously. It is characterised by a raised annular configuration.
- No common sequelae are recognised, although rarely it may involve underlying fascia or tendon sheaths.
- Some evidence exists of aberrant cellular-mediated immune response in adults, but these studies have not been replicated in children. No treatment has been shown categorically to alter the course of the lesion. The most obvious differential would be necrobiosis lipoidica diabetorum.

Skin disorders associated with malignancy

Paraneoplastic syndromes associated with internal malignancies:

Skin disorder	Associated malignancies
Tylosis	Oesophageal cancer
Acanthosis nigricans	Gastric cancer- GI cancer
Migratory thrombophlebitis	Pancreatic cancer
Necrolytic migratory erythema	Glucagonoma
	
Acquired hypertrichosis lanuginosa	Gastrointestinal and lung cancer
Dermatomyositis	Ovarian and lung cancer
Erythema gyratum repens	Lung cancer
Pyoderma gangrenosum (bullous and non-bullous forms)	Myeloproliferative disorders
Sweet's syndrome also known as acute febrile neutrophilic dermatosis	Haematological malignancy e.g. Myelodysplasia - tender, purple plaques
Acquired ichthyosis	Lymphoma
Erythroderma	Lymphoma

-Lymphoma:----> Erythroderma,Acquired ichthyosis

-Lung cancer -----> Dermatomyositis---Erythema gyratum repens---Acquired hypertrichosis lanuginosa,

-Gastric cancer-----> Acanthosis nigricans

-Oesophageal cancer---> Tylosis

- Pancreatic cancer-----> Migratory thrombophlebitis
- Glucagonoma-----> Necrolytic migratory erythema
- Ovarian-----> Dermatomyositis---
- Haematological malignancy (e.g.Myelodysplasia) ----- >Sweet's syndrome
- Myeloproliferative disorders-----> Pyoderma gangrenosum

Systemic mastocytosis

Systemic mastocytosis results from a neoplastic proliferation of mast cells

Features:

- 1) Urticaria pigmentosa - produces a wheal on rubbing (Darier's sign)
- 2) Flushing
- 3) Abdominal pain
- 4) Monocytosis on the blood film

Diagnosis:

- 1) Raised serum **tryptase** levels
 - 2) Urinary **histamine**
-

Zinc deficiency

Features:

- 1) Perioral dermatitis: red, crusted lesions
- 2) Acrodermatitis
- 3) Alopecia
- 4) Short stature
- 5) Hypogonadism
- 6) Hepatosplenomegaly
- 7) Geophagia (ingesting clay/soil)
- 8) Cognitive impairment

Vitamin B2 (riboflavin) deficiency may also cause angular cheilosis.

Scurvy is a result of vitamin C deficiency. It is required for collagen synthesis. The finding shown is peri-follicular haemorrhage. Other findings include bleeding gums, corkscrew hair and poor wound healing. High-dose vitamin C replacement will usually lead to rapid resolution of symptoms in a few days.

Beri-beri is a result of deficiency of thiamine (vitamin B1). It can present with neurological, cardiovascular and gastrointestinal symptoms.

Pellagra is a result of niacin deficiency. It can present with clinical symptoms and signs starting with the four Ds:

- Dermatitis (photodistributed)
- Dementia
- Diarrhoea, and
- Death.

Vitamin A deficiency presents with ophthalmological problems of night blindness and can eventually lead to blindness if not treated. It can be associated with dry skin and dry hair.

Hypersensitivity

The Gell and Coombs classification divides hypersensitivity reactions into 4 types

Type I – Anaphylactic:

- Antigen reacts with **ige bound to mast cells**
- Anaphylaxis, atopy (e.g. Asthma, eczema and hayfever)

Type II - Cell bound:

- **igg or igm** binds to antigen on cell surface
- **Autoimmune haemolytic anaemia, ITP,**
- **Acute hemolytic transfusion reactions,**
- **Goodpasture's,**
- **Pernicious anemia,**
- **Rheumatic fever,**
- **Bullous pemphigoid, pemphigus vulgaris**

Type III - Immune complex

- Free antigen and antibody (igg, iga) combine
- **Serum sickness,**
- **Systemic lupus erythematosus,**
- **Post-streptococcal glomerulonephritis,**
- **Extrinsic allergic alveolitis (especially acute phase)**

Type IV - Delayed hypersensitivity

- **T cell mediated**
- **Tuberculosis, tuberculin skin reaction,**
- **Graft versus host disease,**
- **Allergic contact dermatitis,**
- **Scabies,**
- **Extrinsic allergic alveolitis (especially chronic phase),**
- **Multiple sclerosis, Guillain-Barre syndrome**

Type V:

- Antibodies that recognise and bind to the cell surface receptors, either stimulating them or blocking ligand binding
- **Graves' disease, myasthenia gravis**

Lichen simplex chronicus (LSC)

- Presents with hyperpigmented, scaly, lichenified plaques.
- Patients may volunteer a history of chronic scratching or manipulation, especially during times of stress.
- The ankles are common sites for LSC.



Pseudoxanthoma elasticum

- Inherited condition (usually autosomal recessive*)
- The disease is rare and is characterised by progressive calcification and degeneration of elastic fibres and
- Primarily affects **the skin, retina and cardiovascular system**.
- Cutaneous manifestations: Small, yellow papules of 1-5 mm in diameter are seen and may coalesce to form plaques.
- The skin takes on a plucked chicken, Moroccan leather, or cobblestone appearance. The skin becomes soft, lax and wrinkled and may hang in folds.
- Extracutaneous manifestations include gastrointestinal haemorrhage, cardiovascular disease (hypertension, accelerated atherosclerosis), angioid streaks in the retina and retinal haemorrhage.

Features:

- 1) Retinal angioid streaks
- 2) **'Plucked chicken skin'** appearance - small yellow papules on the neck, antecubital fossa and axillae
- 3) Cardiac: due to loss of elastic tissue
 - Mitral valve prolapse, MR,
 - AR, Aortic dissection ()
 - Increased risk of ischaemic heart disease & peripheral arterial disease
- 4) Gastrointestinal haemorrhage

*there are reports of autosomal dominant inheritance in a minority of cases



Mycosis fungoides (a cutaneous T cell lymphoma)



- The disease presents as a pruritic eczematous rash (the pre-malignant stage) and develops telangiectasias and areas of 'cigarette paper' atrophy.
 - As malignancy develops, nodular lesions appear and proceed to become necrotic.
-

Botulinum toxin type A (or trade name Botox®) acts by decreasing transmission of acetylcholine across the neuromuscular junction in striated muscles, so inducing a temporary flaccid paralysis in the muscle into which it is injected.

Wrist and hand disability due to upper limb spasticity associated with stroke in adults, and use in cases of muscle spasticity, such as cases of cerebral palsy are well recognised indications for the use of Botox®.

Though not licensed, Botox has been used in other medical conditions, including:

- Primary axillary hyperhidrosis
 - Blepharospasm
 - Strabismus, and
 - Cervical dystonia.
-

Immunofluorescence studies will reveal iga deposits within blood vessel walls in Henoch-Schönlein purpura

Intercellular igg deposits are found in the pemphigoid group of blistering disorders.

Intercellular iga deposits are found in the iga subset of pemphigus.

Fibrin deposits at the basement membrane zone are found in cases of lichen planus (LP), although immunofluorescence studies are uncommonly done to diagnose LP.

Granular iga deposits at the basement membrane zone are found in dermatitis herpetiformis.

- **Macule:** An area of altered skin colour irrespective of the size.
- **Plaque:** a descriptive term for a skin lesion that is raised and greater than 1 cm in diameter.
- **Papule:** a raised lesion less than 1 cm in diameter.
- **An ulcer:** is a discontinuity of the skin with complete loss of the epidermis and often portions of the dermis and subcutaneous fat.
- **A vesicle** is a fluid-filled, well-circumscribed raised lesion.
- **Pustules** are small elevations of the skin containing cloudy or purulent material, usually consisting of necrotic inflammatory cells.
- **Bullae** are large vesicles containing serous fluid.
- **Fissures** are cracks in the skin that are narrow but deep.
- **Telangiectasias** are collections of enlarged capillaries visible on the skin or mucous membranes
- **Lichenification** of the skin is due to epidermal thickening characterised by visible and palpable thickening of the skin with accentuation of skin markings.
- **Atrophy of the skin** may be due to loss of epidermis, dermis or subcutaneous tissue. Thinning of the epidermis presents as skin that appears thin and translucent. Thinning of the dermis and subcutaneous tissue leads to a depression in the skin.

Polymorphous light eruption:

- Presents most commonly in young females.
- Various morphologies have been described, for example, macules, papules, patches or plaques. However, in a given patient, the rash usually appears the same whenever it recurs.
- It commonly occurs a few hours after sun-exposure and resolves after a few days, without scarring.

Solar urticaria

- Presents with urticaria upon sun-exposure.
- This usually resolves in a few hours instead of a few days.

Acute lupus erythematosus

- May present with a similar rash to polymorphous light eruption. However, patients will usually have other symptoms like joint pains, oral ulcers, hair loss, etc.
- They frequently have positive anti-nuclear antibodies.

Discoid lupus erythematosus

- Presents with chronic scaly, erythematous-to-hyperpigmented papules and plaques that leave residual scars and dyspigmentation.
- The rash is worsened by sun-exposure.

Xeroderma pigmentosum

- Patients present with exquisite sun sensitivity from a very young age.
- They also present with multiple solar lentigines, nevi and malignant skin tumours, for example, basal cell carcinoma, squamous cell carcinoma and malignant melanoma.